The heritability and genetics of individual variation in human colour vision are well understood, with molecular and physiological mechanisms in the retina particularly well characterised. In contrast, very little is known about the heritability and mechanisms of individual variation for any post-retinal visual processing phenomena. Binocular rivalry (BR) is a well-studied perceptual phenomenon that occurs when dissimilar images are presented in corresponding locations of the two eyes. To resolve the resulting visual ambiguity, perception alternates every few seconds between the conflicting images, at a rate that is relatively stable within individuals but that varies widely between individuals. The determinants and mechanisms of individual variation in BR rate have yet to be elucidated. Here, using a large genetically informative sample, we present evidence demonstrating a substantial genetic contribution to an individual’s BR rate. One hundred and twenty-eight monozygotic (MZ) and 220 dizygotic (DZ) twin pairs, and 26 unpaired co-twins, reported BR with orthogonal drifting gratings, over 21 minutes of viewing. Correlations for BR rate in MZ and DZ twins were 0.51 and 0.19, respectively. Genetic modelling showed 52% of the variance in BR rate was accounted for by additive genetic factors in the best fitting model. This is the first study to report a substantial genetic contribution to individual variation in BR rate, and furthermore, is the first large study to do so for any post-retinal visual processing phenomena. The results have important implications for understanding BR mechanisms, and suggest that genetic and molecular approaches to investigating the phenomenon should be vigorously pursued. The results also have important clinical and pathophysiological implications because BR rate is abnormally slow in bipolar disorder, a common psychiatric condition known to be highly heritable.

Much has been learned about the heritability and physiology of colour vision since Thomas Young, Hermann von Helmholtz and Ewald Hering proposed theories of colour perception over a century ago. In addition to competing colour vision theories, Helmholtz and Hering proposed competing theories of BR (Fig. 1a), suggesting top-down and bottom-up models, respectively. As with their theories on colour vision, their approaches to BR are today both considered relevant. Thus, many studies of BR have favoured low-level explanations of the phenomenon, while many others have favoured high-level models. The current consensus is that BR involves a series of neural processes at different levels of the visual pathway. Despite this consensus, a detailed mechanistic understanding of processing at each level, and of interactions between levels, has yet to be achieved.

One aspect of BR that has been extensively studied is its temporal dynamics. Several extrinsic (exogenous) factors during BR are known to determine the relative strength of one stimulus over its rival (i.e. predominance), and to also determine an individual’s BR rate. These include, for example, the contrast, spatial frequency, velocity and semantic context of the stimuli. However, far less is known about the intrinsic (endogenous) factors that determine an individual’s BR rate when stimulus and ambient conditions are held constant. The present study aimed to investigate whether there is a genetic contribution to individual variation in BR rate.

Our interest in this aspect of BR was motivated by a relative lack of focus on individual differences in BR research and a lack of BR models addressing such differences. We were also motivated by previous data suggesting slow BR may serve as an endophenotype for bipolar disorder (manic depression), a psychiatric condition with a lifetime prevalence of 1–5% and reported heritability of 0.67–0.85. A key feature of a putative endophenotype is that it should be a heritable trait. To examine the heritability of BR, we utilised the twin method and studied both MZ twins (who are genetically identical) and DZ twins (who share roughly half of their genes), aged 14 years. The twin method enables parsing of the familial similarities in a trait into genetic and shared environmental sources, with the remaining variance attributed to unique environmental factors, including measurement errors (Fig. 1c). We report a substantial genetic contribution to individual variation in BR rate.
Fig. 1 | a. Presenting a different image to each eye simultaneously in the same retinal location induces BR – perceptual alternation between each image every few seconds (with occasional mixed periods). The speed at which this perceptual alternation occurs is referred to as BR rate and this parameter is known to vary between individuals.4,5.  
b. BR data were collected in 3 blocks of four trials over half an hour, with interspersed breaks. The first block was used for training and to allow BR rates to stabilise (Methods). This block was discarded before analysis and BR parameters were examined for block 2 and 3 data only, with mixed percepts excluded. c. Independent Pathway modelling of variance into additive (A) and non-additive (i.e., dominance/epistasis, D) genetic sources, unique (E) environmental sources, and measurement error/unreliability (U). Reliable genetic and environmental variance is identified by equating pathways from A, D, and E factors to first and second test occasion data. The remaining variance (U) is unshared between the two test occasions but represents an equal amount of variance for each variable on each test occasion and is therefore equated. Correlations between co-twins for A and D factors are fixed.

Exclusive BR (i.e., the absence of mixed percepts) was achieved with the employed stimuli as indicated by the low third-response-option (TRO) hits and associated time (Table 1; Methods Summary). BR rates varied widely between individuals, by an order of magnitude, and normalised predominance values also varied between individuals (Table 1). The distribution of normalised BR interval durations was well described by a gamma function (Fig. 2a). The maximum likelihood within-test reliability for BR rate (Fig. 2b) and for TRO hits/time was very high. For predominance, this reliability measure was lower, but still high (Table 1). Reliability over time (retest) was high for BR rate (Fig. 2c) and moderate for predominance and TRO hits/time (Table 1). Measures of between-block change in predominance showed poor reliability over time (retest correlations non-significant) and therefore are not reported.

The main finding is significantly higher BR rate correlation in MZ twins (0.51) compared with DZ twins (0.19), thus indicating genetic influence on this parameter (Fig. 3). In contrast, the MZ and DZ twin correlations for predominance and TRO hits/time were not significant, so these measures were not included in genetic modelling analyses. Preliminary analyses of BR rate prior to genetic modelling showed homogeneity of sampling, with no birth order, zygosity, or sex effects for means or variances. Further, no mean effect was found for age. However, a significant mean effect was found for acuity (Δχ² = 17.0), such that 37 individuals with acuity of 6/9 in either eye had a marginally slower BR rate (0.45Hz) than the rest of the sample (0.55Hz) for whom acuity was 6/6 or better in both eyes. Twin correlations were rerun on only those participants with equal visual acuity in each twin pair, and the results did not differ from those obtained for the full sample.

There was no evidence of common environmental influence (i.e. DZr was not > 0.5*MZr; Fig. 3). Rather, there was a suggestion of non-additive genetic influence. Therefore, BR rate was examined in a model allowing for additive (A) and non-additive (D) genetic influences, unique environmental influences (E), and measurement unreliability (U). Estimates for A, E, and U were significant, with A accounting for 45% of the variance (95% confidence intervals 24, 62), E accounting for 19% (3, 35), and U accounting for 30% (22, 42). D was not significant, accounting for 6% (0, 22). Consequently, an AEU model (Fig. 3) was the most parsimonious and the best-fitting model with 52% of the variance attributed to genetic influence.

The present study represents the largest BR population dataset yet published, and thus provides large-scale confirmation of previous reports that BR rate varies widely between individuals. The data also confirm previous reports of high retest reliability of BR rate and lower retest reliability of BR predominance. Despite being collected from participants aged only 14 years, the present BR rate results can be considered highly reliable because of the very high correlation between individuals’ block 2 and block 3 data. Indeed, this is consistent with within-test reliability data previously shown for adults. Thus, in the present study, high within-test reliability, the gamma analysis results (also consistent with adult BR data), and the wide range of BR rates, show that BR at age 14 is not importantly different from BR in adults. The data also show that there is no mean change in BR rate from the age of 14 to 16 years. This study therefore adds to the limited developmental and lifespan studies of BR.

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Table 1. Summary statistics, reliability correlations and twin correlations of BR data

<table>
<thead>
<tr>
<th>Test</th>
<th>Mean</th>
<th>Log Predominance</th>
<th>TRO</th>
<th>ρTRO</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean</td>
<td>0.5</td>
<td>0.17</td>
<td>18.4</td>
<td>33.9</td>
</tr>
<tr>
<td>Range</td>
<td>0.11</td>
<td>0.09</td>
<td>0.18</td>
<td>0.23</td>
</tr>
<tr>
<td>Reliability: Within-test r (95% CI)</td>
<td>0.79</td>
<td>0.43</td>
<td>0.39</td>
<td>0.29</td>
</tr>
</tbody>
</table>

| Reliability: Between-test r (95% CI) | 0.75 | 0.56 | 0.78 | 0.23 | 0.59 | 0.03 | 0.53 | 0.13 | 0.00 |

| Twin correlations: MZ r (95% CI) | 0.51 | 0.06 | 0.08 | 0.10 | 0.02 | 0.03 | 0.03 | 0.03 |

Note: TRO (third response option) indicates mixed, unequal and uncertain percepts, or an response not shown in (SD) and range for hits and time associated with vertical and horizontal perceiving percepts. These tables respectively are hit and set xed.  
All hits were categorised for correlation analysis due to non-normal distribution (thresholds were set that divided each variable into three categories of approximately equal size based on scores from the first test occasion). Not shown are the group mean, (SD) and range for individuals mean and median percent correct. These values respectively are 2.05 (0.96, 0.79) and 1.60 (0.71, 0.74, 0.67). Twin correlations for individuals’ mean and median perception durations were not significantly different from those for BR rate. All mean, (SD) and range values shown are before twinization. Prior to reliability and twin correlation analyses, data were transformed to z = 3 SD.

The present study represents the largest BR population dataset yet published, and thus provides large-scale confirmation of previous reports that BR rate varies widely between individuals. The data also confirm previous reports of high retest reliability of BR rate and lower retest reliability of BR predominance. Despite being collected from participants aged only 14 years, the present BR rate results can be considered highly reliable because of the very high correlation between individuals’ block 2 and block 3 data. Indeed, this is consistent with within-test reliability data previously shown for adults. Thus, in the present study, high within-test reliability, the gamma analysis results (also consistent with adult BR data), and the wide range of BR rates, show that BR at age 14 is not importantly different from BR in adults. The data also show that there is no mean change in BR rate from the age of 14 to 16 years. This study therefore adds to the limited developmental and lifespan studies of BR.  

Normalised distribution of interval durations. The gamma function that specifies values to generate a gamma curve of best fit to the data and suggests novel approaches to studying BR mechanisms. The following gamma function parameters are reported: $N = \text{total number of intervals}$, $n = \text{number of subjects}$, $\varphi = \text{mean interval duration (seconds)}$ across all subjects, $\sigma = \text{standard deviation of all intervals}$, $R^2 = \text{coefficient of determination}$. $\lambda$ and $\sigma$ are parameters that specify values to generate a gamma curve of best fit to the normalised distribution of interval durations. The gamma function equation used: $f(x) = \frac{\lambda^r}{\Gamma(r)} x^{r-1} \exp(-\lambda x)$ where $\Gamma(r) = (r-1)!$.

We have demonstrated, for the first time, a substantial genetic contribution to individual variation in BR rate. Indeed, this is the first such finding in a large sample for any post-retinal visual processing phenomenon. While there have been previous reports of genetic contributions to illusory movement, flicker fusion thresholds, and Rorschach indices, the sample sizes examined were small. A recent large twin study assessed contrast sensitivity in middle-aged males and found a modest heritability estimate (0.14–0.38), however it is not known whether deficient contrast sensitivity occurs in the lens, retina, or during post-retinal processing. Inspection time for line-length discrimination has also been examined in a large twin sample and been shown to have substantial genetic influence (heritability estimate, 0.57), but this perceptual task is thought to reflect attentional and decision processes used in response monitoring. While some authors have proposed a role for decision-making during BR, it is also well-known that the perceptual alternations cannot be prevented and voluntary attention has only a limited effect on the phenomenon.

In contrast to visual processing phenomena, twin and family studies have been much more widely applied to the investigation of cognitive and higher cortical functions, as well as various central nervous system disorders. Thus, genetic contributions have been reported for individual variation in attentional function, working memory, affective regulation, personality, and intelligence, as well as a variety of neuropsychiatric disorders. The emerging genetics of attentional networks may have particular relevance to linking the findings of the present study to underlying BR mechanisms. This is because although voluntary attention has only a limited effect on the phenomenon, mechanisms of involuntary attention are thought to be engaged during BR. Indeed, the present study paves the way for further detailed investigation of genetic and molecular aspects of BR, and suggests novel approaches to studying BR mechanisms.

Proposed mechanistic models of BR have included rivalry between monocular channel neurons in primary visual cortex, rivalry between stimulus representations at a high level of visual processing, hierarchical computational models, and rivalry between independent attentional selection mechanisms in each cerebral hemisphere. The methods used to investigate proposed models include electrophysiological studies in animals, and in humans, psychophysical, electrophysiological, magnetoencephalographic, functional magnetic resonance imaging, and brain stimulation studies. Perception-dependent neural activity during BR has been reported as early as lateral geniculate nucleus, in primary visual cortex, as late as inferotemporal cortex and also non-visual regions. Similarly, modulation of BR can occur with brain stimulation applied at low and high levels. Despite evidence of multi-level processing involved in BR, specific mechanisms within and between levels, remain unclear.

Recent BR studies have thus been largely focused on the level at which the phenomenon occurs in the brain, and have generally failed to account for individual variation in BR rate. Similarly, few BR studies have examined the role of neurotransmitter systems and the effects of pharmacological agents, though recent reports suggest involvement of serotonergic and noradrenergic systems. Such studies may provide clues to the molecular mechanisms of individual variation in BR rate, and indeed this issue should now be vigorously pursued given the present findings. One model of BR, which remains under investigation, has suggested that cationic channel levels within a subcortical switch mechanism determine individual variation in BR rate. Those authors also reported that BR rate was significantly slower in subjects with bipolar disorder than in control subjects, a finding that has since been replicated. The slowing of BR in

![Fig. 2](image_url) a. The frequency histogram of normalised interval durations is well-described by the gamma curve given the high coefficient of determination value ($R^2 = 0.99$). This function also describes adult BR interval durations. In the distribution each subject's individual perceptual interval durations are normalised to the subject's mean percept duration (with vertical and horizontal grating percepts pooled). The following gamma function parameters are reported: $N = \text{total number of intervals}$, $n = \text{number of subjects}$, $\varphi = \text{mean interval duration (seconds)}$ across all subjects, $\sigma = \text{standard deviation of all intervals}$, $R^2 = \text{coefficient of determination}$. $\lambda$ and $\sigma$ are parameters that specify values to generate a gamma curve of best fit to the normalised distribution of interval durations. The gamma function equation used: $f(x) = \frac{\lambda^r}{\Gamma(r)} x^{r-1} \exp(-\lambda x)$ where $\Gamma(r) = (r-1)!$. b. Scatterplot showing very high within-test (block 2 vs block 3) reliability of BR rate in 14 year-old twins (N=722). c. Scatterplot showing high between-test (retest) reliability of BR rate in 14 year-old twins retested at age 16 years (N=97).

In contrast to visual processing phenomena, twin and family studies have been much more widely applied to the investigation of cognitive and higher cortical functions, as well as various central nervous system disorders. Thus, genetic contributions have been reported for individual variation in attentional function, working memory, affective regulation, personality, and intelligence, as well as a variety of neuropsychiatric disorders. The emerging genetics of attentional networks may have particular relevance to linking the findings of the present study to underlying BR mechanisms. This is because although voluntary attention has only a limited effect on the phenomenon, mechanisms of involuntary attention are thought to be engaged during BR. Indeed, the present study paves the way for further detailed investigation of genetic and molecular aspects of BR, and suggests novel approaches to studying BR mechanisms.

![Fig. 3](image_url) | MZ and DZ twin correlations for BR rate (left) and associated genetic modelling analyses (right) of this BR parameter in an AEU (best-fitting) model. The model estimates the proportions of variance influenced by additive genes (52%), unique environment (18%), and measurement unreliability over a period of 2 years (30%). Examination of within-test reliability indicated that only 7% of the total variance (23% of the unreliable variance shown above) is due to within-test unreliability.
bipolar disorder also draws empirical support from similar findings with other types of perceptual rivalry and, and the fact that these other rivalry types have properties in common with BR, and may share some degree of common neural mechanisms.

The present finding of a genetic contribution to individual variation in BR rate supports previous suggestions that slow BR may serve as a useful endophenotype for bipolar disorder, because putative endophenotypes for heritable conditions must themselves be heritable traits. Given previous reports that BR rate but not predominance is abnormal in bipolar disorder, we therefore expected the present findings that BR rate but not predominance would be heritable. Our demonstration that BR rate is a heritable quantitative trait has the potential to reveal not only mechanisms of BR, but also mechanisms of bipolar disorder. Indeed, it suggests further research involving gene-finding strategies for both BR rate and bipolar disorder. It also suggests further characterisation of slow BR as a putative bipolar endophenotype, and use of this trait in genetic research to overcome challenges posed by heterogeneity of the bipolar clinical phenotype.

Finally, we propose that as for colour vision and the retina, BR may serve as a paradigm case to unravel the genetics, physiology and pathophysiology of post-retinal vision and perception. Although BR is likely to be a complex trait with complex inheritance (unlike colour vision), it is a phenomenon that has proven highly amenable to research in animals and humans using a wide variety of investigative methods. It is a phenomenon that may also provide a unique window into the science, and indeed genetics, of visual consciousness.

### METHODS SUMMARY

BR was assessed in a large, genetically informative sample (N=722; 48% male; 128 MZ pairs, 220 DZ pairs, and 26 unpaired co-twins; mean age=14.1±0.1SD, 14-15 years). A small sub-sample (N=97; 53% male) was retested 1.9 to 2.8 years (M = 2.1±0.2) after the first test. Zygosity was determined by typing 9 independent polymorphic DNA markers using the AmpFLSTR® Profiler® PCR Amplification Kit and crosschecked with ABO, MN and Rh blood groups and/or phenotypic information (hair, skin and eye colour). Based on this, zygosity was assigned with an extremely low probability of error (less than 10^-3). BR stimuli comprised drifting vertical and horizontal square-wave gratings, viewed through LCD goggles, with no training in fixation required. Participants pressed one raised key (left hand) to indicate perception of the vertical grating, an adjacent raised key (right hand) for the horizontal grating, and the TRO for mixed, unusual or uncertain percepts, or to indicate a previously incorrect response. TRos were excluded. BR data were collected for 21 minutes in 3 blocks of 4 trials with interspersed breaks. The analyses included data from blocks 2 and 3 only, and were performed with specialized software (BiReme Systems) and PASW Statistics 17.0. The primary measures were BR rate (Hz), predominance (ratio of time spent viewing one image relative to the other), number of TRO hits and time associated with TROs. Within- and between-test reliability and genetic analyses used the structural equation modelling package Mx which utilises the method of maximum likelihood estimation from raw data observations. Means and variances were examined for birth order, zygosity, and sex effects, and further, means were examined for age and acuity effects. Genetic modelling is shown in Fig. 1c and modelling results in Fig. 3.

### METHODS

#### Participants

A population sample of twins was recruited by the Queensland Institute of Medical Research for a genetic study of melanocytic naevi (moles) through mail-outs to schools in South-East Queensland between 2000 and 2009. BR data were collected during a routine mole-count visit scheduled when twins turned 14 years of age. At approximately 16 years of age, twins were invited to participate in a study of cognition and a subset was retested for BR. The retest sample comprised 11 MZ pairs, 35 DZ pairs, and 5 unpaired co-twins aged 16.0 to 16.9 years (M = 16.1±0.2SD). Individuals at age 14 or 16 were excluded if they (a) reported a history of, or medication for, depression or ADHD, (b) reported a history of brain injury or other neurological condition, (c) reported a history of uncorrected strabismus, (d) had visual acuity worse than 6/9 in either eye (acuity was measured using a Snellen chart at 3 metres), or (e) there were obvious problems with data collection. This led to a total of 26 exclusions at the first test (aged 14) and 11 exclusions at retest (aged 16). Written, informed consent was obtained from all participants and a parent or guardian. The study conformed to the National Statement on Ethical Conduct in Human Research (2007) issued by the National Health and Medical Research Council (NHMRC) of Australia, and was approved by the Queensland Institute of Medical Research Ethics Committee.

#### BR stimuli, recording procedure and measures

Stimuli were presented on a monochrome (green) computer monitor situated 3 metres from the participant, in a dimly lit room. The orthogonal BR stimuli were vertical gratings drifting horizontally left-to-right, always presented to the left eye, and horizontal gratings drifting vertically downwards, always presented to the right eye. The gratings had a spatial frequency of 8 cycles/degree, were drifting at 4 cycles/second and were presented in a circular patch subtending 1.5 degrees of visual angle. Contrast of the gratings was 0.9. The procedure for BR data collection (Fig. 1b) has been outlined in detail elsewhere. The only difference in the recording procedure in the present study was that participants were provided with an explanatory sheet to assist with training, showing the various possible perceptions and explaining how to respond in each scenario. Participants were instructed to view the stimuli passively rather than attempting to influence their perceptions. They were supervised by a research assistant at all times during data collection. Block 1 recording was used to train the subject, checking they understood the instructions and were performing the task correctly. Questions were able to be asked during this period. Block 1 data was discarded before analysis. Block 2 and block 3 data were of most interest because BR rates tend to stabilise with viewing time. BR rate was calculated by dividing the number of perceptual switches by the total viewing period in blocks 2 and 3, excluding the periods immediately preceding and following a TRO. The resulting BR rate value is the number of perceptual switches per second (expressed in Hertz). Predominance was calculated by dividing the total time spent perceiving the vertical grating by the total time spent perceiving the horizontal grating in blocks 2 and 3 (with similar TRO exclusions). The resulting ratio was then log transformed. Although the TRO exclusion meant the remaining data represented exclusive BR periods, the number of TRos and the time associated with TRos indicates more than just non-exclusive BR (mixed percepts) because the TRO was also used to indicate incorrect or undecided responses.
Genetic model fitting. Genetic analysis methods are presented in common
environment (C) or non-additive (D) genetic effects (i.e. dominance/epistasis), but not both, as they are confounded in
twins reared together. Model choice is influenced by the twin
correlations, which indicate the influence of either C (D_{Zr} > .5*M_{Zr}) or D (D_{Zr} < .5*M_{Zr})\textsuperscript{13}. In addition, twin
correlations were examined to see if data could be pooled
across sex. That is, could correlations be set equal for (a) male
and female MZ pairs, (b) male and female DZ (same-sex)
pairs, and subsequently, (c) DZ same-sex and opposite-sex
pairs. Modified independent pathway modelling\textsuperscript{9} of test and
retest data was used to estimate variance due to measurement
error and trait fluctuations (i.e. unreliable variance). Similarly,
data from the first test occasion were divided into two
collection blocks and modelled to estimate within-test
unreliability.

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Author Contributions

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2. Wilmer JB. (2008). How to use individual differences to
isolate functional organization, biology, and utility of visual
functions; with illustrative proposals for stereopsis. Spat Vis.


interhemispheric switch in bipolar disorder? Proceedings of
the Royal Society of London B: Biological Sciences, 265(1411), 2141–2148.

5. Miller, S. M., Gynther, B. D., Heslop, K. R., Liu, G. B.,
Mitchell, P. B., Ngo, T. T., Pettigrew, J. D., & Geffen, L. B.
Psychological Medicine, 33(4), 683–692.

6. Nagamine M, Yoshino A, Miyazaki M, Takahashi Y,
Nomura S. (2009). Difference in binocular rivalry rate
between patients with bipolar I and bipolar II disorders.
Bipolar Disord. 11(5):539-46.


concept in psychiatry: etymology and strategic intentions. Am

generative studies of twins and families. Dordrecht: Kluwer
Academic Publishers.

10. Logothetis, N. K., Leopold, D. A., & Sheinberg, D. L.
(1996). What is rivalling during binocular rivalry? Nature,
380(6575), 621–624.

binocular rivalry. In D. Alais & R. Blake (Eds.), Binocular


Grant MD, Jacobson KC, Eisen SA, Laudate TM, Kremen
WS. (2007). Genetic influence on contrast sensitivity in

15. Luciano M, Posthuma D, Wright MJ, de Geus EJC, Smith
Perceptual speed does not cause intelligence, and intelligence

16. Fossella J, Sommer T, Fan J, Wu Y, Swanson JM, Pfaff
DW, Posner MI. (2002). Assessing the molecular genetics of attention networks. BMC
Neurosci. 4;3:14.

hemispheres. With a note on the correlates and constitution of
visual consciousness. Brain and Mind, 2(1), 119–149.

18. Ngo TT, Liu GB, Tilley AJ, Pettigrew JD, Miller SM
mechanisms for coherence rivalry and eye rivalry: A meta-

19. Miller, S. M., Liu, G. B., Ngo, T. T., Hooper, G., Rick, S.,
switching mediates perceptual rivalry. Current Biology, 10(7),
383–392.

specific effects of binocular rivalry in the human lateral

transcranial magnetic stimulation on visual rivalry. Journal of
Vision, 7(7): 2, 1–11, http://journalofvision.org/7/7/2/,
doi:10.1167/7.7.2.


Supplementary references (Methods references):
